PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, February 24, 1890.

The President, Dr. H. C. WOOD, in the chair.

Dr. J. CHALMERS DA COSTA read a paper on "Ophthalmic Migraine." (See page 217.)

DISCUSSION.

Dr. H. C. WOOD stated that he had recently been consulted, by a professor in one of the Western colleges, by letter. The patient was thirty-five years old, and had suffered occasionally from attacks, similar to those now present, between fourteen and twenty years of age, the attacks always following extra exertion. From 1876 there had been no attacks until the latter part of 1889. The professor describes these paroxysms as follows:

"The attack begins by imperfect vision; usually there appears a spot in the field of vision. Sometimes this spot appears to dance, at other times it simply obscures a portion of the object seen. If I run the eye along a printed line, as in reading, it appears exactly as if something were being drawn along the line, covering up the words as fast as I pass them. If I attempt to call the words from right to left, I cannot do so rapidly, but must wait for the curtain, as it were, to be drawn aside. This feature of the attack, which I shall designate the 'blind spot' stage, usually lasts twenty or thirty minutes. This is always present. Other features of the attack are sometimes wanting. There is usually a sensation of numbness at some time during the attack. Occasionally it precedes the 'blind spot' stage, but often comes on after the latter has disappeared. Sometimes both features are present at the same time. The numbness is usually in hands and fingers, sometimes in forearm. It is frequently in cheeks and lips and tongue. This numbness is not like that resulting from cold, neither like that from partial paralysis of a member, called 'being asleep;' it resembles the latter more than the former. When this numbness is in face or tongue, there is always a dancing or twitching sensation. This feature varies as to its time of appearance and as to its duration.

"Another feature is the severe pain in the head, or rather in the eye. The pain seems to be in the region of the right eye. This pain does not begin until about the time the 'blind spot' stage disappears. The pain lasts some three or four hours. I feel quite well then until the next attack. It may be well to say that I now take five grains of antipyrine when I feel the attack coming on, and

thus avoid much of the pain in the eye. Twice, in the last ten attacks, I did not take the antipyrine immediately, and the severe pain in the eye on those two occasions shows that the almost absence of pain on the other eight occasions was due to its use. As well as I can remember, I have had about fifteen of these attacks within the last four or five weeks."

In subsequent letter Professor ——— stated that he had discovered that his mother had suffered from attacks precisely similar to his own, even to the numbness and imperfection of vision.

There are one or two points worthy of note in regard to these cases. In the first place, as to the theories suggested to account for the disturbance. I think that at present any one has a right to believe any theory he chooses, for all our theories are houses built of cards, as we have not sufficient data to enable us to formulate a positive theory. We can simply say that megrim is of the nature of a nerve-storm, and that this nerve-storm sometimes involves various sensory areas, and more rarely even the motor areas.

I think that in this country severe ophthalmic disturbances are infrequent. I can remember but one pronounced case of ophthalmic megrim; possibly, when there is much disturbance of vision, the cases go to the oculist, rather than to the neurologist.

Many years ago Trousseau alleged that there is a relationship between megrim and epilepsy. I have seen many cases of epilepsy and many cases of megrim, but I have never yet seen a family in which megrim alternated in successive generations with epilepsy. I have seen only one case of megrim in an epileptic, and in that instance there seemed to be no relation between the paroxysms of epilepsy and those of megrim. There may, however, be a form of megrim which is epileptic megrim, just as you may have epileptic gastralgia and epileptic sensory discharges in other parts of the body, replacing the ordinary epileptic motor discharges. But the rare occurrence of such cases is no more proof of relationship between epilepsy and ordinary megrim than is the occurrence of epileptic gastralgia evidence of a relation between epilepsy and ordinary stomach-ache.

Dr. JAMES HENDRIE LLOYD.—I know very well a patient who has been the subject of ophthalmic megrim since childhood. The symptoms are similar to those referred to by Dr. Wood's case. The vision becomes blurred and indistinct, the idea of a curtain before a part of the field of vision

being rather a happy expression of this. This passes off in ten or fifteen minutes, and is replaced by severe headache, unless, before that develops, the patient obtains relief by saline cathartics. I have associated the occurrence of these attacks with gastric disturbance, although usually the patient is not sick at the stomach until after the storm has taken place. These attacks recur periodically two or three times a year. In regard to the hereditary nature of this affection, I believe that one of the patient's grandfathers suffered with it.

Dr. J. MADISON TAYLOR.—I too might add a word of personal experience. Myself and a large number of my relatives suffer from what seems a true migraine—headaches of regularly periodical recurrence, and seemingly unrelated to accidental causes, always excepting worry or nervous strain. These begin in childhood, reach a climax of severity in early adult life, and then happily lessen, both in frequence and severity, till they sometimes cease.

In certain instances it is strictly one-sided, and often localizable in one spot. It is almost always accompanied by increased pain on using the eyes, but no marked visual disturbances. It is interesting to note and satisfactory for me to report that neither in the score or so of cases thus reviewed, nor in the remainder of a pretty large connection, is there any instance of true nervous disorder, except here and there a little neuralgia, nor indeed of any of the diathetic diseases.

The paroxysms are accompanied by coldness of the extremities, with seemingly increased heat of head. The one drug which most efficiently relieves, as I have discovered after much search, is atropia, especially used hypodermatically. The instant the peripheral arterioles begin to relax their spasm, under the benign influence of this rapidly acting drug, the heart quiets down, pain lessens, and, along with this, the often more distressing nervous depression and restlessness.

I hazard the conjecture that, whatever be the *fons et origo* of this disorder, it is usually complicated by a certain amount of intestinal disturbance and fæcal reabsorption. The phenomena of aparoxysm closely resembles ptomaine poisoning, and the treatment suitable for each is much the same.

Dr. H. C. WOOD.—I have noticed in at least one case that during a paroxysm of megrim the jewelry about the person became distinctly tarnished by a sulphurous emanation from the skin: a suggestive fact in connection with the question of ptomaines.

Dr. G. E. DE SCHWEINITZ.—I have had the opportunity of studying a number of cases of ordinary migraine, and some examples of typical ophthalmic migraine. The most remarkable series of hemicranias which have come under my observation, and which should probably be classed as ophthalmic, are those recorded by Dr. Weir Mitchell and myself, in which the prodromal visual disturbance took the form of an apparition. As these cases have already been published in detail, I shall not now refer to them further.

In a certain number of my cases of migraine, hemianopsia, numbness of the mouth, and numbness of the left upper extremity preceded the attack. In one such case I made examinations of the eyegrounds during the paroxysm, without, however, observing phenomena worthy of special record. In two of these cases ascending doses of cannabis indica, pushed to the point of tolerance, afforded the greatest relief. Another curious symptom that I have noted, in addition to the depression which follows intense pain, is that during and after the attack a mental state, amounting to melancholia, appears. In one such case after the paroxysm the patient is in a condition in which he fears that he either has done or will do some great wrong. I do not know whether this is the result of depression from great pain, or whether it is part of the nerve-explosion. As Dr. Wood has said, you may build as many theories in regard to the cause of migraine as you choose, but they fall like houses of cards. One theory, which has been much dwelt upon in modern times, is that all forms of migraine depend upon imperfect ocular balance and disturbances of vision. That these frequently are the origin of a host of violent headaches no one can deny, but that they alone are the cause of the complex phenomena which make up an attack of ophthalmic migraine, or indeed of ordinary migraine, is at least doubtful. It should be remembered that patients the subjects of migraines sometimes have two headaches: one, the hemicrania, as in a case recently seen with Dr. Weir Mitchell, in which the paroxysms were preceded by numbness and scintillating scotomata, and in which general treatment is efficient; and a second, usually persistent, frontal or occipital headache, the result of eye-strain or insufficient ocular muscles, and which is cured by the appropriate ocular therapeutics. I do not believe that the various forms of heterophoria, or refraction-error, cause true ophthalmic migraine, nor have I ever seen a case of this kind cured by their correction alone, however much this may have aided in the favorable result. I have investigated a number of

cases of migraine and epilepsy, and have never found any association, nor have they seemed to have had a common cause.

HYSTERICAL AMBLYOPIA IN A COLORED GIRL NINE YEARS OF AGE.

Dr. G. E. DE SCHWEINITZ.—I wish to report a case of amblyopia of hysterical origin occurring in a colored girl nine years of age. The child was brought to the dispensary of the Hospital of the University of Pennsylvania because she claimed that the left eye was sightless. The right eye was slightly myopic, otherwise healthy, and with the correcting lens the vision rose to normal. In the left eye there was a perfectly normal oval optic disc, healthy in color, and the retina natural. The refraction was hypermetropic. The child denied even light-perception, although the pupil reacted normally to the changes of light and shade. Remembering the observation of Bernutz, that the conjunctiva in hysterical cases is frequently anæsthetic, I tested this in the child under discussion, and found it as insensitive as if the eye had been cocainized. A further examination revealed complete left hemianæsthesia. The ordinary tests with prisms, as well as that one proposed by Dr. Harlan, of this city, readily demonstrated the presence of vision in the eye in which light-perception was denied. The child had had no disease except measles; the mother, a mulatto, was healthy. The treatment consisted in the administration of some water colored with the compound tincture of cardamom, care being taken to impress upon the parent as well as the child the importance of giving exactly the dose, the impression being conveyed that the medicine was most powerful in its action.

The case gradually improved, and now, after a number of months, vision almost normal in amount has returned to the eye for which previously blindness had been claimed. It was practically impossible to make any determination of the color-fields of the right eye, or of the left eye, since the patient has admitted a return of vision.

It is not always easy to classify correctly that form of blindness which is called hysterical, because, to quote the able paper of Dr. Hill-Griffith, "it is sometimes doubtful, as some one has remarked, if the subjects should be considered as patients or as culprits." I should like to hear the opinion of the members of the Society on this point, and on any other points which may help to explain this curious form of amblyopia.

I have made the following interesting experiment in these cases, namely, in one case of simulated blindness (malingering) the subject was made to understand, by having the tests explained to her, that those present at the examination fully understood that her statements in regard to a lack of sight in the eye under examination were totally false. She was sufficiently intelligent to appreciate that she had been detected in her attempt at deceit, and readily admitted the charge. On the other hand, a perfectly intelligent woman, the subject of hysterical amblyopia, in whom the tests demonstrated the fact that the eye claimed for blindness had full visual acuity, utterly denied the possibility of sight, in spite of the fact of her apparent perfect appreciation of the sufficiency of the tests which had demonstrated that she could see.

Dr. CHARLES K. MILLS reported a case of

EMBOLISM OF THE RADIAL ARTERY.

The following case was seen in consultation with Dr. J. J. Healy: The patient, fifty-two years old, had symptoms of hepatic and pulmonary congestion. In about three weeks, when he had nearly recovered, but while still in bed, he was taken suddenly with intense pain in the front of the arm between the median line and the radial border, about two inches below the elbow. The pain extended to the forearm, hand, and fingers, which became practically helpless. All the fingers, with the exception of the little finger, were blue and cold, as were also, but to a less extent, the entire hand and forearm upward to the site of the initial pain. The greatest pain, blueness, and coldness were in the thumb and middle finger; they were like "dead fingers," and were almost completely paralyzed. All movements of the fingers, hand, and wrist, but particularly those of flexion, were much impaired, and slight swelling was present, but no real œdema.

In about twenty-four hours the lividity had considerably abated, and in forty-eight hours circulation was fairly reestablished. The patient, however, continued to suffer greatly and was in a highly nervous state.

He was first seen by me ten days after the onset. A small swelling could be felt at the position of the first pain; the radial pulse on this side had disappeared, and the closed, cord-like vessel could be easily traced. He had great pain on pressure, following the line of the vessel, and considerable but less hyperæsthesia over the entire radial half

of the forearm; as the ulnar border was approached, the pain and hyperæsthesia diminished and disappeared. He complained greatly of feelings of coldness and pain here, and also in the thumb and fingers, except the little finger. Gradually the circulation was more fully restored, and the pain and weakness subsided. At the time of my last visit to the patient, four weeks after the occurrence of the embolism, he still complained of some coldness, tingling and pain in the hand, especially in the thumb, which continued to be more paretic than any other part. The treatment employed was chiefly local warmth, counter-irritation, internally anodynes and tonics. For the present stage, massage has been recommended.

The points which seem most worthy of consideration by a neurologist are the cause and characteristics of the pain, the nature of the paralysis, and the proper treatment. Embolism of the cerebral arteries apparently causes but little pain, doubtless because of the absence of nerves in the cerebral substance. In the extremities, and largely elsewhere, nerves, sensory or mixed, are in close relation with the arteries; the radial nerve, for instance, closely apposed, lies to the outer side of the artery. The persisting pain and hyperæsthesia in these cases are much as in a true neuritis.

DISCUSSION.

Dr. WHARTON SINKLER.—Some years ago I had the opportunity of seeing a lady who had several attacks of embolism of vessels, the result of extreme disease of the mitral valve. The first attack in which I saw her occurred while she had her arm elevated combing her hair. The right arm suddenly dropped motionless; there was violent pain in the arm; the limb was cold, and there was no pulse at the wrist or at the bend of the elbow. The power of motion returned on the following day; the radial pulse did not return.

Some months later she had embolism of one of the cerebral vessels, giving rise to temporary right hemiplegia and aphasia. Later there was another attack, but I do not recall the vessel involved. The first attack occurred some six years ago, and the patient died only a few days ago; but I do not know what was the cause of death, as she died in another city.

Dr. WHARTON SINKLER reported "A Case of Pericarditis occurring during an Attack of Acute Chorea in a Child Nine Years of Age."